dysplasia, glomerular hypertrophy, tubular hypertrophy, glomerulosclerosis and tubulointerstitial sclerosis.

55. (Amended) A method as in claim 2, wherein said chronic renal condition is selected from the group consisting of chronic diabetic nephropathy, diabetic glomerulopathy, diabetic renal hypertrophy, hypertensive nephrosclerosis, hypertensive glomerulosclerosis, renal dysplasia, glomerular hypertrophy, tubular hypertrophy, glomerulosclerosis and tubulointerstitial sclerosis.

REMARKS

In the Office Action of March 15, 2001, claims 1-4, 6-10, 12, 15-17, 24, 28, 32 and 52-55 have been provisionally rejected as under the judicially created doctrine of obviousness-type double patenting as being unpatentable over the claims of copending U.S. Application No. 08/643321.

Applicants reiterate that they are prepared to file a terminal disclaimer to effectively overcome this ground of rejection pending notification of allowable subject in this application.

Claims 1-4, 6-0, 12, 15-17, 24, 28, 32 and 52-55 have been rejected under 35 U.S.C. 112, first paragraph, as not being enabled by the specification. The Examiner states that while the claims are enabled for a method of improving renal function, or delaying the need for dialysis in a mammal wherein said mammal is afflicted with a specific renal condition, the specification does not provide enablement for a method of improving renal function or delaying the need for dialysis in a mammal afflicted with a non-immune, non-inflammatory condition.

In response, the claims have now been amended to delete the phrase "non-immune, noninflammatory condition". This phrase has been replaced with language reciting that the condition is a chronic renal condition characterized by the progressive loss of renal function associated with the progressive loss of functioning nephron units. Antecedent support for this language is found in the specification at page 4, lines 11-17, as well on page 11, lines 5-13. A chronic condition characterized by the progressive loss of renal function is also, by implication, a noninflammatory condition. Thus, there is ample literal support for the present claim language, and the rejection has been effectively obviated.

Claims 1 and 2 have also been rejected under 35 U.S.C., second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter of the invention. In particular, the claims have been objected to for reciting a non-immune, noninflammatory condition.

The language which has been objected to by the Examiner has now been changed by applicants. Accordingly, the basis for this rejection has been rendered moot.

In view of the foregoing facts and reasons, the claims of this application are now deemed to overcome the remaining grounds of rejection, and to fulfill all remaining requirements for patentability. Entry of the foregoing amendment is deemed appropriate at this time since it merely results in a simplification of the issues, and does not require any further search or consideration. Accordingly, entry of this amendment, at least for purposes of appeal, and allowance of the remaining claim in this application is solicited.

Respectfully submitted,

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MARKED-UP CLAIMS

1. (Four Times Amended) A method of improving renal function in a mammal in, or at risk of, chronic renal failure comprising administering to said mammal a therapeutically effective amount of a morphogen, said morphogen comprising an amino acid sequence having at least 70% amino acid sequence homology with the C-terminal seven-cysteine skeleton of human OP-1, amino acids 330-431 of SEQ ID NO:1;

wherein said mammal is not a kidney transplant recipient, and is afflicted with a chronic renal [non-immune, noninflammatory] condition characterized by the progressive loss of renal function associated with the progressive loss of functioning nephron units; wherein said morphogen induces chondrogenesis in an *in vivo* ectopic bone assay; and wherein said therapeutically effective amount causes a clinically significant improvement in a standard marker of renal function in said mammal.

2. (Four Times Amended) A method of treatment to delay the need for, or reduce the frequency of, chronic dialysis treatments in a mammal in, or at risk of, chronic renal failure comprising

administering to said mammal a therapeutically effective amount of a morphogen, said morphogen comprising an amino acid sequence having at least 70% amino acid sequence homology with the C-terminal seven-cysteine skeleton of human OP-1, amino acids 330-431 of SEQ ID NO:1;

wherein said mammal is not a kidney transplant recipient, and is afflicted with a chronic renal [non-immune. noninflammatory] condition characterized by the progressive loss of renal function associated with the progressive loss of functioning nephron units; wherein said morphogen induces chondrogenesis in an *in vivo* ectopic bone assay; and wherein said therapeutically effective amount causes a clinically significant improvement in a

standard marker of renal function in said mammal such that said mammal's need for chronic dialysis is delayed or reduced.

54. (Amended) A method as in claim 1, wherein said [non-immune, noninflammatory] <u>chronic renal</u> condition is selected from the group consisting of: chronic diabetic nephropathy, diabetic glomerulopathy, diabetic renal hypertrophy, hypertensive nephrosclerosis, hypertensive glomerulosclerosis, renal dysplasia, glomerular hypertrophy, tubular hypertrophy, glomerulosclerosis and tubulointerstitial sclerosis.

55. (Amended) A method as in claim 2, wherein said [non-immune, noninflammatory] <u>chronic renal</u> condition is selected from the group consisting of: chronic diabetic nephropathy, diabetic glomerulopathy, diabetic renal hypertrophy, hypertensive nephrosclerosis, hypertensive glomerulosclerosis, renal dysplasia, glomerular hypertrophy, tubular hypertrophy, glomerulosclerosis and tubulointerstitial sclerosis.